Kwon
PCT/10685

```
=> fil medl,biosis,embase,caplus,wpids
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 12:46:38 ON 16 NOV 2005

FILE 'BIOSIS' ENTERED AT 12:46:38 ON 16 NOV 2005
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FILE 'EMBASE' ENTERED AT 12:46:38 ON 16 NOV 2005
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FILE 'CAPLUS' ENTERED AT 12:46:38 ON 16 NOV 2005
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FILE 'WPIDS' ENTERED AT 12:46:38 ON 16 NOV 2005
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=> s (mouse or mice)(w)(p53)(l)strain(l)(129 or sv trp5n)
L1             2 FILE MEDLINE
L2             2 FILE BIOSIS
L3             1 FILE EMBASE
L4             2 FILE CAPLUS
L5             0 FILE WPIDS

TOTAL FOR ALL FILES
L6             7 (MOUSE OR MICE)(W)(P53)(L) STRAIN(L)(129 OR SV TRP5N)

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7             2 DUP REM L6 (5 DUPLICATES REMOVED)

=> d 1-2 ibib abs hit

L7   ANSWER 1 OF 2     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    96001391     MEDLINE
DOCUMENT NUMBER:     PubMed ID: 7546219
TITLE:               Effects of genetic background on tumorigenesis in
                     p53-deficient mice.
AUTHOR:              Donehower L A; Harvey M; Vogel H; McArthur M J; Montgomery
                     C A Jr; Park S H; Thompson T; Ford R J; Bradley A
CORPORATE SOURCE:    Division of Molecular Virology, Baylor College of Medicine,
                     Houston, TX 77030, USA.
CONTRACT NUMBER:     CA16672 (NCI)
     CA50588 (NCI)
     CA54897 (NCI)
SOURCE:              Molecular carcinogenesis, (1995 Sep) 14 (1) 16-22.
                     Journal code: 8811105. ISSN: 0899-1987.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199511
ENTRY DATE:          Entered STN: 19951227
                     Last Updated on STN: 19951227
                     Entered Medline: 19951106
AB   Mice with disrupted germline p53 alleles have been engineered by us and
```

others and have been shown to have enhanced susceptibility to spontaneous tumors of various types. We monitored a large number of p53-deficient **mice** (**p53**+/- and p53-/-) and their wild-type littermates (p53+/+) of two different genetic backgrounds (**129**/Sv and mixed C57BL/6 x **129**/Sv) up to 2 yr of age. p53+/- and p53-/- **129**/Sv mice show accelerated tumorigenesis rates compared with their p53-deficient counterparts of mixed C57BL/6 x **129**/Sv genetic background. The tumor spectra of the two **strains** of mice are similar except that almost half of **129**/Sv p53-/- males develop malignant teratomas, whereas these tumors are rarely observed in C57BL/6 x **129**/Sv mice and never in **129**/Sv p53+/- males. In the study reported here, we further characterized the lymphomas that arose in the p53-nullizygous mice and found that over three-quarters of the lymphomas were of thymic origin and contained primarily immature (CD4+/CD8+) T-cells, whereas the remainder originated in the spleen and peripheral lymph nodes and were of B-cell type. The high incidence of early-onset lymphomas in the nullizygous mice makes these animals a good lymphoma model, whereas the heterozygous mice may be a useful model for Li-Fraumeni syndrome, a human inherited cancer predisposition.

AB Mice with disrupted germline p53 alleles have been engineered by us and others and have been shown to have enhanced susceptibility to spontaneous tumors of various types. We monitored a large number of p53-deficient **mice** (**p53**+/- and p53-/-) and their wild-type littermates (p53+/+) of two different genetic backgrounds (**129**/Sv and mixed C57BL/6 x **129**/Sv) up to 2 yr of age. p53+/- and p53-/- **129**/Sv mice show accelerated tumorigenesis rates compared with their p53-deficient counterparts of mixed C57BL/6 x **129**/Sv genetic background. The tumor spectra of the two **strains** of mice are similar except that almost half of **129**/Sv p53-/- males develop malignant teratomas, whereas these tumors are rarely observed in C57BL/6 x **129**/Sv mice and never in **129**/Sv p53+/- males. In the study reported here, we further characterized the lymphomas that arose in the p53-nullizygous mice and found that over three-quarters of the lymphomas were of thymic origin and contained primarily immature (CD4+/CD8+) T-cells, whereas the remainder originated in the spleen and peripheral lymph nodes and were of B-cell type. The high incidence of early-onset lymphomas in the nullizygous mice makes these animals a good lymphoma model, whereas the heterozygous mice may be a useful model for Li-Fraumeni syndrome, a human inherited cancer predisposition.

L7 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 95129816 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7828835
TITLE: Homology of p53 intronic sequences between four laboratory mouse strains and Japanese wild mouse (Mus musculus molossinus Mishima).
AUTHOR: Tokumitsu M; Ogawa K
CORPORATE SOURCE: Department of Pathology, Asahikawa Medical College, Japan.
SOURCE: Genome / National Research Council Canada = Genome / Conseil national de recherches Canada, (1994 Dec) 37 (6) 1022-6.
Journal code: 8704544. ISSN: 0831-2796.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950307
Last Updated on STN: 19950307
Entered Medline: 19950223

AB **Strain** variation in the **mouse p53** gene sequences was investigated in various regions of the gene in 14 inbred **strains** of laboratory mice and one Japanese wild mouse **strain** (Mus musculus molossinus Mishima, M. MOL-MSM). Nucleotides within p53 introns 1 and 7, found to be identical in 10 of the laboratory **strains** (**129**/J, A/J, AKR/J, BALB/cJ, C3H/HeJ, C57BL/6J, CBA/J, CE/J, NZB, and SWR/J), were substituted for other nucleotide sequences in common with M. MOL-MSM and the four other **strains** (DBA/1J, DBA/2J, I/LnJ, and P/J). The latter were documented to have originated from a common ancestor. These observations thus suggested the possibility that the p53 gene may have become substituted by outcrossing of this ancestral **strain** with Asian mice; this is presumably related to the documentation that Japanese mice brought to western countries were used as laboratory mice early in this century. To establish p53 gene heterozygosity, female C3H/HeJ and male DBA/2J mice were mated to produce F1 hybrids (C3D2F1). Electrophoresis of PCR fragments including polymorphic regions with or without restriction enzyme digestion, allowed clear distinction of paternal and maternal p53 alleles. These markers, therefore, should be useful for studying the loss of heterozygosity of the p53 gene during the carcinogenic process.

AB **Strain** variation in the **mouse p53** gene sequences was investigated in various regions of the gene in 14 inbred **strains** of laboratory mice and one Japanese wild mouse **strain** (Mus musculus molossinus Mishima, M. MOL-MSM). Nucleotides within p53 introns 1 and 7, found to be identical in 10 of the laboratory **strains** (**129**/J, A/J, AKR/J, BALB/cJ, C3H/HeJ, C57BL/6J, CBA/J, CE/J, NZB, and SWR/J), were substituted for other nucleotide sequences in common with M. MOL-MSM and the four other **strains** (DBA/1J, DBA/2J, I/LnJ, and P/J). The latter were documented to have originated from a common ancestor. These observations thus suggested the possibility that the p53 gene may have become substituted by outcrossing of this ancestral **strain** with Asian mice; this is presumably related to the documentation that Japanese mice brought to western countries were used as laboratory mice early in this century. To establish p53 gene heterozygosity, female C3H/HeJ and male DBA/2J mice were mated to produce F1 hybrids (C3D2F1). Electrophoresis of PCR fragments including polymorphic regions with or without restriction enzyme digestion, allowed clear distinction of paternal and maternal p53 alleles. These markers, therefore, should be useful for studying the loss of heterozygosity of the p53 gene during the carcinogenic process.

```
=> s sv(w)trp5n or svtrp5n
L8            0 FILE MEDLINE
L9            0 FILE BIOSIS
L10           0 FILE EMBASE
L11           0 FILE CAPLUS
L12           0 FILE WPIDS

TOTAL FOR ALL FILES
L13           0 SV(W) TRP5N OR SVTRP5N

=> fil reg
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               26.34      26.55

FILE 'REGISTRY' ENTERED AT 12:49:30 ON 16 NOV 2005
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```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 15 NOV 2005 HIGHEST RN 868125-94-4
DICTIONARY FILE UPDATES: 15 NOV 2005 HIGHEST RN 868125-94-4

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2005

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

```
**********************************************************************
*                                                                    *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,    *
* effective March 20, 2005.  A new display format, IDERL, is now     *
* available and contains the CA role and document type information.  *
*                                                                    *
**********************************************************************
```

Structure search iteration limits have been increased. See HELP SLIMITS
for details.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

```
=> e nitroxide/cn 5
E1            1     NITROXAZEPINE/CN
E2            1     NITROXEN/CN
E3            1 --> NITROXIDE/CN
E4            1     NITROXIDE (DISINFECTANT)/CN
E5            1     NITROXIDE ION(1-)/CN

=> s e3
L14           1 NITROXIDE/CN

=> e "4-hydroxy-2,2,6,6-tetramethylpiperidine-1-oxyl"/cn
E1            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE N-OXIDE/CN
E2            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE OXIDE/CN
E3            1 --> 4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-1-OXYL/CN
E4            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-1-OXYL 4-DIHYDROGEN
                    PHOSPHATE/CN
E5            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-1-OXYL BENZOATE/CN
E6            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-4-CARBOXYLIC ACID HY
                    DRAZIDE/CN
E7            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-D17-1-OXYL/CN
E8            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-N-OXY/CN
E9            1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-N-OXYL/CN
E10           1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINIUM N-OXIDE TRIFLATE/C
                    N
E11           1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINO-1-OXYL/CN
E12           1     4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINO-1-OXYL MONOETHYL PHO
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

SPHOROFLUORIDATE ESTER/CN

```
=> s e3;e tempol/cn 5
L15           1 "4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-1-OXYL"/CN


E1            1     TEMPOCHOLINE CHLORIDE/CN
E2            1     TEMPOET/CN
E3            1 --> TEMPOL/CN
E4            1     TEMPOL BENZOATE/CN
E5            1     TEMPOL H/CN

=> s e3;fil medl,biosis,embase,caplus;s l14 or nitroxide;s l15 or
hydroxy(l)tetramethylpiperidine(l)oxyl;s l16 or tempol
L16           1 TEMPOL/CN


COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      15.09      41.64

FILE 'MEDLINE' ENTERED AT 12:51:13 ON 16 NOV 2005

FILE 'BIOSIS' ENTERED AT 12:51:13 ON 16 NOV 2005
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L17        1933 FILE MEDLINE
L18        2273 FILE BIOSIS
L19        1952 FILE EMBASE
L20        9751 FILE CAPLUS

TOTAL FOR ALL FILES
L21       15909 L14 OR NITROXIDE


L22         481 FILE MEDLINE
L23         539 FILE BIOSIS
L24         586 FILE EMBASE
L25        2509 FILE CAPLUS

TOTAL FOR ALL FILES
L26        4115 L15 OR HYDROXY(L) TETRAMETHYLPIPERIDINE(L) OXYL


L27         607 FILE MEDLINE
L28         709 FILE BIOSIS
L29         632 FILE EMBASE
L30        2670 FILE CAPLUS

TOTAL FOR ALL FILES
L31        4618 L16 OR TEMPOL
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
=> s (mouse or mice)(w)p53 and l21 and (l26 or l31)
L32          0 FILE MEDLINE
L33          1 FILE BIOSIS
L34          0 FILE EMBASE
L35          0 FILE CAPLUS

TOTAL FOR ALL FILES
L36          1 (MOUSE OR MICE)(W) P53 AND L21 AND (L26 OR L31)

=> d

L36  ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
AN   2005:352534  BIOSIS
DN   PREV200510137680
TI   Cancer chemoprevention by the antioxidant tempol acts partially
     via the p53 tumor suppressor.
AU   Erker, Laura; Schubert, Ralf; Yakushiji, Hiroyuki; Barlow, Carrolee;
     Larson, Denise; Mitchell, James B.; Wynshaw-Boris, Anthony [Reprint
     Author]
CS   Univ Calif San Diego, Sch Med, Dept Pediat, 9500 Gilman Dr,Mailstop 0627,
     La Jolla, CA 92093 USA
     awynshawboris@ucsd.edu
SO   Human Molecular Genetics, (JUN 15 2005) Vol. 14, No. 12, pp. 1699-1708.
     ISSN: 0964-6906(print).
DT   Article
LA   English
ED   Entered STN: 8 Sep 2005
     Last Updated on STN: 8 Sep 2005


=> e jackson lab/cs
E1           1     JACKSON KOFI ABAKA/CS
E2           1     JACKSON KOFI ABAKA GHANA/CS
E3           0 --> JACKSON LAB/CS
E4           1     JACKSON LAB 600 MAIN ST BAR HARBOR MA 04609 USA/CS
E5           1     JACKSON LAB 600 MAIN ST BAR HARBOR MAIN 04609 0800 USA/CS
E6           5     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE 04609 0800 USA/CS
E7           3     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE 04609 1500 USA/CS
E8          38     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE 04609 USA/CS
E9           1     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE 06409 USA/CS
E10          1     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE USA/CS
E11          1     JACKSON LAB 600 MAIN ST BAR HARBOR MAINE USA 04609 USA/CS
E12          1     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04060 USA/CS

=> s jackson lab?/cs
L37       1243 FILE MEDLINE
TERM 'LAB?' EXCEEDED TRUNCATION LIMITS - SEARCH ENDED
You have entered a truncated stem which occurs in too many terms.
Make the stem longer and try again.  For example, if your original
term was 'degr?' to search for variations and the abbreviation for
'degradation', you could replace it with the expression '(degrdn OR
degrad?)'.  If your search term was numeric, e.g., 'C>5', reduce the
size of the range.

=> s jackson lab ?/cs
ADDITIONAL CHARACTERS REQUIRED AFTER '?' FOR LEFT TRUNCATION
Additional characters must follow the left truncation symbol in your
search term.  If your search term contains a punctuation mark before
the truncation symbol and you are searching in a field that uses
```

implied proximity, the system may interpret the truncation symbol as being at the beginning of a term. Implied proximity is used in search fields indexed as single words, for example, the Basic Index. To see which fields in the current file have left truncation, enter "HELP SFIELDS" at an arrow prompt (=>).

```
=> e
E13          1     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04509 USA/CS
E14          4     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04609 0800 USA/CS
E15         29     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04609 1500 USA/CS
E16        189     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04609 USA/CS
E17          1     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04609 USDA USA/CS
E18          1     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04660 USA/CS
E19          1     JACKSON LAB 600 MAIN ST BAR HARBOR ME 04675 USA/CS
E20          1     JACKSON LAB 600 MAIN ST BAR HARBOR MI 04609 USA/CS
E21          1     JACKSON LAB 600 MAIN ST BAR HARBOR MN 04609 USA/CS
E22          1     JACKSON LAB 600 MAIN ST BOX 258 BAR HARBOR MAINE 04609 USA/C
                   S
E23          1     JACKSON LAB 600 MAIN ST BOX 258 BAR HARBOR ME 04609 USA/CS
E24          1     JACKSON LAB 600 MAIN STREET BAR HARBON ME 04609 USA/CS

=> e
E25          1     JACKSON LAB 600 MAIN STREET BAR HARBOR MA 04609 USA/CS
E26          8     JACKSON LAB 600 MAIN STREET BAR HARBOR MAINE 04609 USA/CS
E27          1     JACKSON LAB 600 MAIN STREET BAR HARBOR ME 04609 0800 USA/CS
E28          3     JACKSON LAB 600 MAIN STREET BAR HARBOR ME 04609 1500 USA/CS
E29          1     JACKSON LAB 600 MAIN STREET BAR HARBOR ME 04609 1600 USA/CS
E30         25     JACKSON LAB 600 MAIN STREET BAR HARBOR ME 04609 USA/CS
E31          1     JACKSON LAB 600 MAINE ST BAR HARBOR MAINE 04609 USA/CS
E32          2     JACKSON LAB 600 MAINE ST BAR HARBOR ME 04609 USA/CS
E33          1     JACKSON LAB 6000 MAIN STREET BAR HARBOR MAINE 04609 1500 USA
                   /CS
E34          1     JACKSON LAB BAR BARBOR ME USA/CS
E35          8     JACKSON LAB BAR HABOR MAINE 04609 USA/CS
E36          1     JACKSON LAB BAR HABOR ME/CS

=> e
E37          2     JACKSON LAB BAR HABOR ME 04609 USA/CS
E38          1     JACKSON LAB BAR HABOR ME USA/CS
E39          1     JACKSON LAB BAR HARBAR ME 04609 USA/CS
E40          1     JACKSON LAB BAR HARBON ME 04609 USA/CS
E41          1     JACKSON LAB BAR HARBOR EDINBURGH UK/CS
E42          1     JACKSON LAB BAR HARBOR MA 02142 USA/CS
E43          6     JACKSON LAB BAR HARBOR MA 04609 USA/CS
E44          3     JACKSON LAB BAR HARBOR MA USA/CS
E45          8     JACKSON LAB BAR HARBOR MAIN 04609 USA/CS
E46          1     JACKSON LAB BAR HARBOR MAIN MAIN/CS
E47          7     JACKSON LAB BAR HARBOR MAINE 04069 USA/CS
E48          3     JACKSON LAB BAR HARBOR MAINE 04609/CS

=> e
E49          3     JACKSON LAB BAR HARBOR MAINE 04609 0800 USA/CS
E50        466     JACKSON LAB BAR HARBOR MAINE 04609 USA/CS
E51          2     JACKSON LAB BAR HARBOR MAINE 04609 USA USA/CS
E52          1     JACKSON LAB BAR HARBOR MAINE 04699 USA/CS
E53          2     JACKSON LAB BAR HARBOR MAINE ME 04609 USA/CS
E54         37     JACKSON LAB BAR HARBOR MAINE USA/CS
E55          1     JACKSON LAB BAR HARBOR MANIE 04609/CS
E56          2     JACKSON LAB BAR HARBOR MASS 04609 USA/CS
E57          1     JACKSON LAB BAR HARBOR MD USA/CS
```

```
E58          44     JACKSON LAB BAR HARBOR ME/CS
E59           1     JACKSON LAB BAR HARBOR ME 04 609 USA/CS
E60           3     JACKSON LAB BAR HARBOR ME 04069 USA/CS

=> e
E61           1     JACKSON LAB BAR HARBOR ME 0460 USA/CS
E62           1     JACKSON LAB BAR HARBOR ME 04604 USA/CS
E63           5     JACKSON LAB BAR HARBOR ME 04609 0800 USA/CS
E64          24     JACKSON LAB BAR HARBOR ME 04609 1500 USA/CS
E65         653     JACKSON LAB BAR HARBOR ME 04609 USA/CS
E66           1     JACKSON LAB BAR HARBOR ME 046092 USA/CS
E67           1     JACKSON LAB BAR HARBOR ME 04679 USA/CS
E68           1     JACKSON LAB BAR HARBOR ME 04909 USA/CS
E69         318     JACKSON LAB BAR HARBOR ME USA/CS
E70           1     JACKSON LAB BAR HARBOR ME USA 04609/CS
E71           2     JACKSON LAB BAR HARBOR MICH 04609 USA/CS
E72           2     JACKSON LAB BAR HARBOR MN 04609 USA/CS

=> e
E73           2     JACKSON LAB BAR HARBOR USA/CS
E74           2     JACKSON LAB BAR HARBOUR MAINE 04609 USA/CS
E75           2     JACKSON LAB BAR HARBOUR ME USA/CS
E76           1     JACKSON LAB BAT HARBOR MAINE USA/CS
E77           1     JACKSON LAB BOX 202 600 MAIN ST BAR HARBOR ME 04609 USA/CS
E78           1     JACKSON LAB BOX 258 600 MAIN ST BAR HARBOR ME 04609 USA/CS
E79           2     JACKSON LAB BOX 258 BAR HARBOR MAINE 04609 USA/CS
E80           1     JACKSON LAB BOX 258 BAR HARBOR ME 04609 USA/CS
E81           1     JACKSON LAB BOX 261 600 MAIN ST BAR HARBOR MA 04609 USA/CS
E82           1     JACKSON LAB BOX 303 600 MAIN ST BAR HARBOR ME 04609 USA/CS
E83           1     JACKSON LAB CHAMBERS WORKS DEEPWATER N J 08023 USA/CS
E84           1     JACKSON LAB CHAMBERS WORKS DEEPWATER NJ 08023 USA/CS

=> s e4-82
L38           0 FILE MEDLINE
L39        1235 FILE BIOSIS
L40           0 FILE EMBASE
L41         723 FILE CAPLUS

TOTAL FOR ALL FILES
L42        1958 ("JACKSON LAB 600 MAIN ST BAR HARBOR MA 04609 USA"/CS OR "JACKSO
                N LAB 600 MAIN ST BAR HARBOR MAIN 04609 0800 USA"/CS OR "JACKSON
                 LAB 600 MAIN ST BAR HARBOR MAINE 04609 0800 USA"/CS OR "JACKSON
                 LAB 600 MAIN ST BAR HARBOR MAINE 04609 1500 USA"/CS OR "JACKSON
                 LAB 600 MAIN ST BAR HARBOR MAINE 04609 USA"/CS OR "JACKSON LAB
                600 MAIN ST BAR HARBOR MAINE 06409 USA"/CS OR "JACKSON LAB 600
                MAIN ST BAR HARBOR MAINE USA"/CS OR "JACKSON LAB 600 MAIN ST
                BAR HARBOR MAINE USA 04609 USA"/CS OR "JACKSON LAB 600 MAIN ST
                BAR HARBOR ME 04060 USA"/CS OR "JACKSON LAB 600 MAIN ST BAR
                HARBOR ME 04509 USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR
                ME 04609 0800 USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR ME
                04609 1500 USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR ME
                04609 USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR ME 04609
                USDA USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR ME 04660
                USA"/CS OR "JACKSON LAB 600 MAIN ST BAR HARBOR ME 04675 USA"/CS
                OR "JACKSON LAB 600 MAIN ST BAR HARBOR MI 04609

=> s l42 and (strain 129 or p53)
L43           0 FILE MEDLINE
L44           9 FILE BIOSIS
L45           0 FILE EMBASE
```

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

L46 4 FILE CAPLUS

TOTAL FOR ALL FILES
L47 13 L42 AND (STRAIN 129 OR P53)

=> dup reml 47
ENTER REMOVE, IDENTIFY, ONLY, OR (?):end

=> dup rem l47
PROCESSING COMPLETED FOR L47
L48 12 DUP REM L47 (1 DUPLICATE REMOVED)

=> d 1-12 ibib abs hit

L48 ANSWER 1 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:465854 BIOSIS
DOCUMENT NUMBER: PREV200400463476
TITLE: An efficient SNP system for mouse genome scanning and elucidating strain relationships.
AUTHOR(S): Petkov, Petko M. [Reprint Author]; Ding, Yueming; Cassell, Megan A.; Zhang, Weidong; Wagner, Gunjan; Sargent, Evelyn E.; Asquith, Steven; Crew, Victor; Johnson, Kevin A.; Robinson, Phil; Scott, Valerie E.; Wiles, Michael V.
CORPORATE SOURCE: **Jackson Lab, 600 Main St, Bar Harbor, ME, 04609, USA**
pmp@jax.org
SOURCE: Genome Research, (September 2004) Vol. 14, No. 9, pp. 1806-1811. print.
ISSN: 1088-9051 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2004
Last Updated on STN: 1 Dec 2004

AB A set of 1638 informative SNP markers easily assayed by the Amplifluor genotyping system were tested in 102 mouse strains, including the majority of the common and wild-derived inbred strains available from The Jackson Laboratory. Selected from publicly available databases, the markers are on average apprx1.5 Mb apart and, whenever possible, represent the rare allele in at least two strains. Amplifluor assays were developed for each marker and performed on two independent DNA samples from each strain. The mean number of polymorphisms between strains was 608+/-136 SD. Several tests indicate that the markers provide an effective system for performing genome scans and quantitative trait loci analyses in all but the most closely related strains. Additionally, the markers revealed several Subtle differences between closely related mouse strains, including the groups of several 129, BALB, C3H, C57, and DBA strains, and a group of wild-derived inbred strains representing several Mus musculus Subspecies. Applying a neighbor-joining method to the data, we constructed a mouse strain family tree, which in most cases confirmed existing genealogies.

CS **Jackson Lab, 600 Main St, Bar Harbor, ME, 04609, USA**
pmp@jax.org

ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
Mus musculus castaneus (subspecies)
Mus musculus domesticus (subspecies)
Mus musculus molossinus (subspecies)
Mus musculus musculus (subspecies)

mouse (common): 102 strains, **strain-129**,
strain-BALB, strain-C3H, strain-C57, strain-DBA
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

L48 ANSWER 2 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:328632 BIOSIS
DOCUMENT NUMBER: PREV200400329472
TITLE: Genetic contributors to lipoprotein cholesterol levels in an intercross of 129S1/SvImJ and RIIIS/J inbred mice.
AUTHOR(S): Lyons, Malcolm A.; Korstanje, Ron; Li, Renhua; Walsh, Kenneth A.; Churchill, Gary A.; Carey, Martin C.; Paigen, Beverly [Reprint Author]
CORPORATE SOURCE: **Jackson Lab, 600 Main St, Bar Harbor, ME, 04609, USA**
bjp@jax.org
SOURCE: Physiological Genomics, (April 13 2004) Vol. 17, No. 2, pp. 114-121. print.
ISSN: 1094-8341 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Jul 2004
Last Updated on STN: 29 Jul 2004

AB To determine the genetic contribution to variation among lipoprotein cholesterol levels, we performed quantitative trait locus (QTL) analyses on an intercross between mouse strains RIIIS/J and 129S1/SvImJ. Male mice of the parental strains and the reciprocal F1 and F2 populations were fed a high-cholesterol, cholic acid-containing diet for 8-12 wk. At the end of the feeding period, plasma total, high-density lipoprotein (HDL), and non-HDL cholesterol were determined. For HDL cholesterol, we identified three significant QTLs on chromosomes (Chrs) 1 (D1Mit507, 88 cM, 72-105 cM, 4.8 LOD), 9 (D11Mit149, 14 cM, 10-25 cM, 9.4 LOD), and 12 (D12Mit60, 20 cM, 0-50 cM, 5.0 LOD). These QTLs were considered identical to QTLs previously named Hdlq5, Hdlq17, and Hdlq18, respectively, in crosses sharing **strain 129.** For total cholesterol, we identified two significant QTLs on Chrs 1 and 9, which were named Chol10 (D1Mit507, 88 cM, 10-105 cM, 3.9 LOD) and Chol11 (D11Mit149, 14 cM, 0-30 cM, 4.4 LOD), respectively. In addition, for total cholesterol, we identified two suggestive QTLs on Chrs 12 (distal) and 17, which remain unnamed. For non-HDL cholesterol, we identified and named one new QTL on Chr 17, Nhdlq3 (D17Mit221, 58 cM, 45-60 cM, 3.4 LOD). Nhdlq3 colocalized with orthologous human QTLs for lipoprotein phenotypes, and with Abcg5 and Abcg8. Overall, we detected eight QTLs for lipoprotein cholesterol concentrations on Chrs 1, 9, 12, and 17 (each two per chromosome), including a new QTL for non-HDL cholesterol, Nhdlq3, on Chr 17.
CS **Jackson Lab, 600 Main St, Bar Harbor, ME, 04609, USA**
bjp@jax.org
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identified two significant QTLs on Chrs 1 and 9, which were named Chol10 (D1Mit507, 88 cM, 10-105 cM, 3.9 LOD) and Chol11 (D11Mit149, 14 cM, 0-30 cM, 4.4 LOD), respectively. In addition, for total cholesterol, we identified two suggestive QTLs on Chrs 12 (distal) and 17, which remain unnamed. For non-HDL cholesterol, we identified and named one new QTL on Chr 17, Nhdlq3 (D17Mit221, 58 cM, 45-60 cM, 3.4 LOD). Nhdlq3 colocalized with orthologous human QTLs for lipoprotein phenotypes, and with Abcg5 and Abcg8. Overall, we detected eight QTLs for lipoprotein cholesterol concentrations on Chrs 1, 9, 12, and 17 (each two per chromosome), including a new QTL for non-HDL cholesterol, Nhdlq3, on Chr 17.

L48 ANSWER 3 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:165418 BIOSIS
DOCUMENT NUMBER: PREV199800165418
TITLE: Mouse strain backgrounds: More than black and white.
AUTHOR(S): Frankel, Wayne N. [Reprint author]
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME 04609, USA**
SOURCE: Neuron, (Feb., 1998) Vol. 20, No. 2, pp. 183. print.
ISSN: 0896-6273.
DOCUMENT TYPE: Letter
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Apr 1998
Last Updated on STN: 6 Apr 1998
CS **Jackson Lab., Bar Harbor, ME 04609, USA**
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Organism Name
mouse: **strain-129**, strain-A, strain-BALB/c, strain-C3H, strain-C57BL/6, strain-CAST, strain-CBA, strain-DBA/2, strain-FVB, strain-NZB, strain-SJL
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals, Rodents, Vertebrates

L48 ANSWER 4 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:241625 BIOSIS
DOCUMENT NUMBER: PREV199800241625
TITLE: Apoptosis in the retinal of tubby mice.
AUTHOR(S): Ikeda, S.; Naggert, J. K.; Nishima, P. M.
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME, USA**
SOURCE: IOVS, (March 15, 1998) Vol. 39, No. 4, pp. S569. print.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. May 10-15, 1998. Association for Research in Vision and Ophthalmology.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jun 1998
Last Updated on STN: 4 Jun 1998
CS **Jackson Lab., Bar Harbor, ME, USA**
IT Major Concepts
Molecular Genetics (Biochemistry and Molecular Biophysics); Sense Organs (Sensory Reception)
IT Parts, Structures, & Systems of Organisms
retina: sensory system, apoptosis
IT Chemicals & Biochemicals

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

p53: mutation, pathway; tub gene: mutation

L48 ANSWER 5 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1994:270588 BIOSIS
DOCUMENT NUMBER: PREV199497283588
TITLE: A mutation in the Ter gene causing increased susceptibility to testicular teratomas maps to mouse chromosome 18.
AUTHOR(S): Asada, Yoshinobu; Varnum, Don S.; Frankel, Wayne N.; Nadeau, Joseph H. [Reprint author]
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME 04609, USA**
SOURCE: Nature Genetics, (1994) Vol. 6, No. 4, pp. 363-368. ISSN: 1061-4036.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jun 1994
Last Updated on STN: 24 Jun 1994
AB Little is known about inherited susceptibility to spontaneous germ cells tumours in humans or other species. The Ter mutation in laboratory mice is novel in that it acts codominantly to reduce germ cell numbers on many inbred strain backgrounds and to enhance dramatically inherited predisposition to spontaneous testicular teratocarcinomas in **strain 129** inbred mice. We have adopted a PCR-based, DNA pooling method for mice with 'extreme' phenotypes (small testes versus normal-sized testes) to identify a candidate linkage to the Ter locus. Two independent mapping approaches confirmed this evidence for Ter linkage near D18Mit62 on mouse chromosome 18, and suggest a possible human homologue on chromosome 5q.
CS **Jackson Lab., Bar Harbor, ME 04609, USA**
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L48 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:320717 CAPLUS
DOCUMENT NUMBER: 120:320717
TITLE: A mutation in the Ter gene causing increased susceptibility to testicular teratomas maps to mouse chromosome 18
AUTHOR(S): Asada, Yoshinobu; Varnum, Don S.; Frankel, Wayne N.; Nadeau, Joseph H.
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME, 04609, USA**
SOURCE: Nature Genetics (1994), 6(4), 263-8
CODEN: NGENEC; ISSN: 1061-4036
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Little is known about inherited susceptibility to spontaneous germ cells tumors in humans or other species. The Ter mutation in laboratory mice is novel in that it acts codominantly to reduce germ cell nos. on many inbred strain backgrounds and to enhance dramatically inherited predisposition to spontaneous testicular teratocarcinomas in **strain 129** inbred mice. The authors have adopted a PCR-based, DNA pooling method for

mice with extreme phenotypes (small testes vs. normal-sized testes) to identify a candidate linkage to the Ter locus. Two independent mapping approaches confirmed this evidence for Ter linkage near D18Mit62 on mouse chromosome 18, and suggest a possible human homolog on chromosome 5q.

CS **Jackson Lab., Bar Harbor, ME, 04609, USA**

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L48 ANSWER 7 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 1990:236149 BIOSIS
DOCUMENT NUMBER: PREV199089123102; BA89:123102
TITLE: ATHEROSCLEROSIS SUSCEPTIBILITY DIFFERENCES AMONG PROGENITORS OF RECOMBINANT INBRED STRAINS OF MICE.
AUTHOR(S): PAIGEN B [Reprint author]; ISHIDA B Y; VERSTUYFT J; WINTERS R B; ALBEE D
CORPORATE SOURCE: **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**
SOURCE: Arteriosclerosis, (1990) Vol. 10, No. 2, pp. 316-323. CODEN: ARTRDW. ISSN: 0276-5047.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 19 May 1990
Last Updated on STN: 19 May 1990

AB Female mice of 16 inbred mouse strains were fed an atherogenic diet for 14 weeks and were then evaluated for atherosclerotic lesions in the aorta. Strains C57BL/6, C57BR/cd, C57L, and SM were very susceptible to atherosclerosis, with lesion area/aortic cross-sections in the range of 4500 to 8000 μm2. Strains C58 and SWR were intermediate in susceptibility, with lesion area/sections in the range of 1670 to 1690 μm2. **Strains 129**, AKR, DBA/2, and BALB/c had only small lesions in the range of 20 to 350 μm2/section; strains C3H, NZB, CBA, HRS, SJL, and A had no lesions after 14 weeks. Lesion formation in five strains was compared at several time points. Strain C57BL/6 mice developed lesions by 7 weeks, and these continued to grow until all mice had large atheromatous plaques in the aorta and coronary arteries. Strains AKR and DBA/2 also had fatty streak lesions as early as 7 or 8 weeks, but these lesions had not progressed in size by 14 weeks. Strains BALB/c and C3H, which were both resistant to lesion formation at 14 weeks, diverged from each other as time progressed. By 1 year, BALB/c mice had large lesions, but C3H mice had none. Most of the inbred strains chosen for evaluation are the progenitors of recombinant inbred sets of strains, a genetic tool that greatly facilitates the analysis of strain differences. This surgery indicates seven additional recombinant inbred sets of strains whose progenitors differ in atherosclerosis susceptibility: BXD, AKXL, SWXJ, NX8, 129XB, NXSM, and B6NXAKRN. An analysis of these recombinant inbred strains may reveal additional mouse genes affecting atherosclerosis susceptibility.

CS **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**

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L48 ANSWER 8 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 1982:283202 BIOSIS
DOCUMENT NUMBER: PREV198274055682; BA74:55682
TITLE: DIETARY MODULATION OF ALPHA CELL VOLUME AND FUNCTION IN **STRAIN 129**-J MICE.
AUTHOR(S): MORLEY M G [Reprint author]; LEITER E H; EISENSTEIN A B; STRACK I
CORPORATE SOURCE: **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**
SOURCE: American Journal of Physiology, (1982) Vol. 242, No. 4, pp. G354-G359.
CODEN: AJPHAP. ISSN: 0002-9513.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB Weanling female 129/J mice were maintained for 1, 2, 3 or 6 mo. on either a control diet containing 60% sucrose and 23% protein or an isocaloric, high-protein, no-carbohydrate diet containing 83% protein and 0% sucrose. Mice were killed after each interval to assess the effect of diet on histological and physiological changes in the endocrine pancreas. Image analysis of islets stained immunocytochemically for α-, β-, δ- and PP cells was performed to quantify changes in islet structure. Islet composition was strongly affected by diet. The volume density of the α-cells was significantly elevated in mice fed the high-protein diet (e.g., 35% vs. 16% in controls at 6 mo.), whereas the volume density of β-cells concomitantly decreased from 65 to 39%. Radioimmunoassay of the insulin and glucagon content of the pancreas and the plasma corroborated the morphometric findings. Pancreatic and plasma glucagon concentration in mice on the high-protein diet was elevated by an average of 2.5-fold above controls, whereas pancreatic insulin concentration was diminished by nearly half. The increase in α-cell volume density and pancreatic glucagon concentration appeared initially due to α-cell hypertrophy, although by 6 mo. of high-protein feeding both hypertrophy and hyperplasia of the α-cells were evident. Presumably, these changes were compensatory responses to the increased functional demand on α-cells (i.e., glucagon biosynthesis and secretion) imposed by chronic high-protein feeding.
TI DIETARY MODULATION OF ALPHA CELL VOLUME AND FUNCTION IN **STRAIN**

129-J MICE.
CS **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**

L48 ANSWER 9 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1980:285631 BIOSIS
DOCUMENT NUMBER: PREV198070078127; BA70:78127
TITLE: A NEW MUTATION DB-3J AT THE DIABETES LOCUS IN **STRAIN 129**-J MICE 2. STUDIES OF PANCREATIC ALPHA CELL FUNCTION IN CULTURE.
AUTHOR(S): LEITER E H [Reprint author]; STRACK I; EISENSTEIN A B
CORPORATE SOURCE: **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**
SOURCE: Diabetologia, (1980) Vol. 19, No. 1, pp. 66-73.
CODEN: DBTGAJ. ISSN: 0012-186X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB Monolayer cell cultures from pancreatic islets of aging 129/J strain diabetes (db3J/db3J) and lean littermate control mice were tested for differences in glucagon and insulin secretion in either serum-free Eagle's minimal essential medium (MEM) or Dulbecco's modified minimal essential medium (DMEM). There was a highly significant ($P < 0.0001$) main effect of genotype and type of culture medium on glucagon secretion with time. Although numbers of A-cells were not demonstrably increased in db3J/db3J cultures in DMEM, mean medium glucagon levels increased 2.7-, 18- and 32-fold above littermate normal culture levels at days 4, 6 and 8, respectively. In MEM, the 2 populations could not be discriminated on the basis of glucagon secretion. Insulin secretion over culture days showed a highly significant ($P < 0.0001$) dependence on genotype, but not type of medium, with the B-cell enriched db3J/db3J preparations secreting between 20 and 30 times as much insulin as controls in both media. Analysis revealed that the heightened secretory responsiveness of mutant A-cells in DMEM as compared to MEM was primarily elicited by the elevated DMEM amino acid concentration and specifically lysine (0.8 mmol/l in DMEM vs. 0.4 mmol/l in MEM). In pulse-chase experiments using 14 day db3J/db3J cultures, incorporation of 3H-tryptophan into protein that eluted from Biogel P-10 columns in the native glucagon peak indicates that DMEM stimulated glucagon biosynthesis as well as secretion. An augmented sensitivity of db3J/db3J A-cells to stimulation by basic amino acids in long-term culture is revealed.
TI A NEW MUTATION DB-3J AT THE DIABETES LOCUS IN **STRAIN 129** -J MICE 2. STUDIES OF PANCREATIC ALPHA CELL FUNCTION IN CULTURE.
CS **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**

L48 ANSWER 10 OF 12 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1981:139793 BIOSIS
DOCUMENT NUMBER: PREV198171009785; BA71:9785
TITLE: A NEW MUTATION DB-3 J AT THE DIABETES LOCUS IN **STRAIN 129**-J MICE 1. PHYSIOLOGICAL AND HISTOLOGICAL CHARACTERIZATION.
AUTHOR(S): LEITER E H [Reprint author]; COLEMAN D L; EISENSTEIN A B; STRACK I
CORPORATE SOURCE: **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**
SOURCE: Diabetologia, (1980) Vol. 19, No. 1, pp. 58-65.
CODEN: DBTGAJ. ISSN: 0012-186X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
AB A spontaneous recessive mutation appearing in **strain 129** /J mice at the diabetes (db) locus on chromosome 4 was characterized. The

new allele, designated db31, produced hyperphagia and severe obesity. Mutants weighed in excess of 70 g by 6 mo. of age, compared to 22-28 g for lean littermates. Although the disease was similar to the mild hyperglycemia-severe obesity syndrome exhibited by db gene presentation on the C57BL/6J inbred background, the syndrome in 129/J mice reduced lifespan, with mutants exhibiting sudden weight loss, hypoglycemia, and a 67% mortality between 6 and 14 mo. of age. Mutant males, but not females, were transiently hyperglycemia between 2-4 mo. of age, attaining a maximum mean blood sugar of 196 ± 27 (standard error of the mean) mg/dl. Thereafter glucose levels declined to normoglycemic values (80-100 mg/dl), and with increasing age, mutants of both sexes became hypoglycemic (60 mg/dl at 9 mo.) . Mutants of both sexes were extremely hyperinsulinemic at the earlier ages, with mean plasma insulin at month 5 reflecting 30-fold elevations above normal for males and 18-fold for females. These levels diminished with age, the decline being more marked in males. Plasma glucagon levels were 3-fold elevated in the younger mutants of both sexes (86 vs. 28 pg/ml in normal mice), mean levels increasing to .apprx. 5-fold above mean control values in the older age group (198 vs. 41 pg/ml in normal mice). Histopathological findings were limited to pancreas. Increasing necrosis of the exocrine, but not endocrine, pancreas was noted in aging mutants. Aldehyde fuchsin staining of the mutant pancreas revealed hyperplastic islets filled with heavily granulated B-cells. B-cell hyperplasia was accompanied by a 30-fold increase over controls in pancreatic insulin content in the 8 mo. old mutants, whereas pancreatic glucagon content was only doubled. Morphometric analysis showed less than a 2-fold increase in the mean number of A-cells per islet. An interesting feature of expression of the diabetes gene in the 129/J strain is the persisting hyperglucagonemia in the face of moderating hyperinsulinemia.

TI A NEW MUTATION DB-3 J AT THE DIABETES LOCUS IN **STRAIN 129**-J MICE 1. PHYSIOLOGICAL AND HISTOLOGICAL CHARACTERIZATION.

CS **JACKSON LAB, BAR HARBOR, MAINE 04609, USA**

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persisting hyperglucagonemia in the face of moderating hyperinsulinemia.

L48 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1967:452512 CAPLUS
DOCUMENT NUMBER: 67:52512
TITLE: Effect of 5-fluorouracil on early teratomas in mice
AUTHOR(S): Aldrich, John T.; Stevens, Leroy Carlton
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME, USA**
SOURCE: Cancer Research (1967), 27(5), 945-9
CODEN: CNREA8; ISSN: 0008-5472
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of 5-fluorouracil (FU) on the development of early teratomas was studied. Testicular teratomas were exptl. induced in **strain 129**/Sv mice by grating genital ridges from 12-day fetuses into the testes of adults. In approx. 80% of the grafts a teratocarcinogenic process was initiated within 24 hrs. The tumors grew and were composed predominantly of neutral tissue. Host mice received a single injection of FU at 50 mg./kg. on one of several days beginning with the day prior to grafting and ending with the 11th day following grafting. Development of teramatous foci was markedly inhibited in grafts in mice treated on days 1-6. Those in hosts treated on days 7-11 had an increasing incidence of tumors approaching that of controls (78%). FU at 25 mg./kg. also prevented the growth of tumors when injected into host mice on day 0 or day 1.
CS **Jackson Lab., Bar Harbor, ME, USA**
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L48 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1966:432817 CAPLUS
DOCUMENT NUMBER: 65:32817
ORIGINAL REFERENCE NO.: 65:6126f-h
TITLE: Polygenic control of the teratogenicity of 5-fluorouracil in mice
AUTHOR(S): Dagg, C. P.; Schlager, Gunther; Doerr, Ann
CORPORATE SOURCE: **Jackson Lab., Bar Harbor, ME**
SOURCE: Genetics (1966), 53(6), 1101-17
CODEN: GENTAE; ISSN: 0016-6731
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The intraperitoneal injection of 5-fluorouracil into pregnant female mice produced higher frequencies of cleft palate and malformed hind leg in the fetuses of inbred mouse **strain 129**/Gg than in strain BALB/cDg. The number of genetic factors involved in the interstrain difference was estimated by genetic studies. It appeared that a min. of 4 loci played a role in determining the incidence of malformed hind feet with a degree of genetic determination of 80%. There was a low but significant correlation between the frequency of malformed hind feet and the body weight

of the mother. Malformed hind feet occurred with nearly equal frequencies in both males and females. Similar estns. for cleft palate gave a min. of 3 loci and a degree of genetic determination of 83%. There was a significant neg. correlation between the body weight of the mother and the incidence of cleft palate. Cleft palate tended to occur slightly more often in female than in male fetuses. Thus, the set of genetic factors influencing the incidence of malformed hind feet in response to 5-fluorouracil were apparently not completely identical with those influencing cleft palate. 20 references.

CS **Jackson Lab., Bar Harbor, ME**

AB The intraperitoneal injection of 5-fluorouracil into pregnant female mice produced higher frequencies of cleft palate and malformed hind leg in the fetuses of inbred mouse **strain 129**/Gg than in strain BALB/cDg. The number of genetic factors involved in the interstrain difference was estimated by genetic studies. It appeared that a min. of 4 loci played a role in determining the incidence of malformed hind feet with a degree of genetic determination of 80%. There was a low but significant correlation between the frequency of malformed hind feet and the body weight of the mother. Malformed hind feet occurred with nearly equal frequencies in both males and females. Similar estns. for cleft palate gave a min. of 3 loci and a degree of genetic determination of 83%. There was a significant neg. correlation between the body weight of the mother and the incidence of cleft palate. Cleft palate tended to occur slightly more often in female than in male fetuses. Thus, the set of genetic factors influencing the incidence of malformed hind feet in response to 5-fluorouracil were apparently not completely identical with those influencing cleft palate. 20 references.

=> dis his all

(FILE 'HOME' ENTERED AT 12:46:03 ON 16 NOV 2005)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, WPIDS' ENTERED AT 12:46:38 ON 16 NOV 2005

L1 2 FILE MEDLINE
L2 2 FILE BIOSIS
L3 1 FILE EMBASE
L4 2 FILE CAPLUS
L5 0 FILE WPIDS
TOTAL FOR ALL FILES
L6 7 S (MOUSE OR MICE)(W)(P53)(L)STRAIN(L)(129 OR SV TRP5N)
L7 2 DUP REM L6 (5 DUPLICATES REMOVED)
L8 0 FILE MEDLINE
L9 0 FILE BIOSIS
L10 0 FILE EMBASE
L11 0 FILE CAPLUS
L12 0 FILE WPIDS
TOTAL FOR ALL FILES
L13 0 S SV(W)TRP5N OR SVTRP5N

FILE 'REGISTRY' ENTERED AT 12:49:30 ON 16 NOV 2005
E NITROXIDE/CN 5
L14 1 S E3
E "4-HYDROXY-2,2,6,6-TETRAMETHYLPIPERIDINE-1-OXYL"/CN
L15 1 S E3
E TEMPOL/CN 5
L16 1 S E3

Prepared by: Mary Hale @2-2507 Rem Bldg 1D86

```
     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 12:51:13 ON 16 NOV 2005
L17         1933 FILE MEDLINE
L18         2273 FILE BIOSIS
L19         1952 FILE EMBASE
L20         9751 FILE CAPLUS
     TOTAL FOR ALL FILES
L21        15909 S L14 OR NITROXIDE
L22          481 FILE MEDLINE
L23          539 FILE BIOSIS
L24          586 FILE EMBASE
L25         2509 FILE CAPLUS
     TOTAL FOR ALL FILES
L26         4115 S L15 OR HYDROXY(L)TETRAMETHYLPIPERIDINE(L)OXYL
L27          607 FILE MEDLINE
L28          709 FILE BIOSIS
L29          632 FILE EMBASE
L30         2670 FILE CAPLUS
     TOTAL FOR ALL FILES
L31         4618 S L16 OR TEMPOL
L32            0 FILE MEDLINE
L33            1 FILE BIOSIS
L34            0 FILE EMBASE
L35            0 FILE CAPLUS
     TOTAL FOR ALL FILES
L36            1 S (MOUSE OR MICE)(W)P53 AND L21 AND (L26 OR L31)
                 E JACKSON LAB/CS
L37         1243 FILE MEDLINE
L38            0 FILE MEDLINE
L39         1235 FILE BIOSIS
L40            0 FILE EMBASE
L41          723 FILE CAPLUS
     TOTAL FOR ALL FILES
L42         1958 S E4-82
L43            0 FILE MEDLINE
L44            9 FILE BIOSIS
L45            0 FILE EMBASE
L46            4 FILE CAPLUS
     TOTAL FOR ALL FILES
L47           13 S L42 AND (STRAIN 129 OR P53)
L48           12 DUP REM L47 (1 DUPLICATE REMOVED)

=> log y
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               206.27     247.91

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)      SINCE FILE      TOTAL
                                                   ENTRY    SESSION
CA SUBSCRIBER PRICE                                -2.19      -2.19

STN INTERNATIONAL LOGOFF AT 12:56:40 ON 16 NOV 2005
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